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Dry foods and risk of disease in cats

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The recent pet food recall seems to have resurrected concerns about the suitability of feeding dry diets to cats. Recalls can be frightening for pet owners and veterinarians alike. One may need to act in the face of significant uncertainty about the extent of the threat, and the stakes, pets' lives, are high. Many clients have a strong emotional bond with their pet and naturally want to do their best for their pet's health and welfare. Since diet plays an important role in pets' well-being, client concerns about pet foods are readily understandable, and their questions deserve honest, empathic responses. The purpose of this paper is to review some of the issues surrounding dry diets for cats.

For a diet to be satisfactory, it must contain all of the necessary nutrients in the proper proportions (complete and balanced), be sufficiently palatable and digestible for the pets consuming it to meet their nutritional needs in the volume consumed, and it must be safe (1). The pet food recall was the result of an inadvertent inclusion of a toxin that made the foods unsafe; there was no evidence that the foods were nutritionally unsatisfactory in any other way. Despite the fact that the recall was for toxicological rather than nutritional reasons, it provided the opportunity for some to question the nutritional adequacy of pet foods.

A commonly raised issue with regard to cat foods is the suitability of carbohydrates in dry foods for cats. Cats are recognized to have evolved as obligate carnivores, consuming foods (small mammals, insects, birds) containing mostly water, protein, and relatively little carbohydrate or fat. Studies have shown that cats are less efficient than some other mammals are at metabolizing dietary carbohydrates under certain circumstances. This observation appears to have led to speculation that long term feeding of carbohydrates may have detrimental effects on the health of cats. Concerns have been raised that some association between the carbohydrate content of dry cat foods and risk of obesity and type 2 diabetes mellitus (DM) may exist, although the relationship, if any, is far from clear. Before attempting to understand these issues, some background may be useful.

Dietary fiber differs from other carbohydrate in the bond configuration between adjacent sugar molecules. Although fibers cannot be degraded by mammalian digestive enzymes, they may be fermented by intestinal microflora depending on their composition and physical properties (fineness of grind). Carbohydrate is not an indispensable nutrient for mammals, although some tissues, such as the brain, red blood cells, and kidneys, prefer glucose as a source of energy. Moreover, carbohydrate is not the only source of glucose. Most amino acids from body or food sources are metabolized to glucose, and small amounts of glucose can be made from the glycerol backbone of triglycerides.

Possibly because of their carnivorous heritage, cats seem to metabolize dietary carbohydrate somewhat differently from other species. As reviewed by Morris (3), cats have an "abridged pattern" of carbohydrate metabolizing enzymes compared with many omnivores, and their pattern of glucose transport enzymes in the liver more closely resembles that of ruminants than other simple-stomached mammals. Both the complexity and processing of dietary carbohydrates may influence its effects on cats; comparable amounts of carbohydrates may have different effects depending on whether they are delivered as simple sugars or starch, and among starches the form of processing may affect carbohydrate utilization. Although cats can efficiently digest cooked starch, they appear to be less tolerant of sucrose (4).

Whether these and other nutritional peculiarities of domestic cats (5) make dry diets unsuitable for them is far from obvious. Although concern is occasionally expressed about feeding "high carbohydrate" diets, the terms "high" and "carbohydrate" are rarely defined clearly. The carbohydrate content of contemporary North American dry cat diets appears to be in the range of 4.5 to 12 g/100 kcal (6). Martin and Rand (7) reported that neutered, sedentary, confined cats consumed approximately 80 kcal/kg body weight (BW)/d (client-owned cats often consume even less than this in my clinical experience), so a 5-kg cat might consume approximately 400 kcal, or about 18 to 48 g of carbohydrate; roughly 4 to 10 g of carbohydrates/kg BW/d. Cats commonly consume small, multiple meals daily. Martin and Rand (7) reported that the cats they studied consumed a median of 9 meals/d; other reports (5) indicated that cats may eat even more frequently, so cats may ingest less than 1 g of carbohydrate/leg BW per meal, even when fed a diet containing the largest quantity of carbohydrate. At this range of carbohydrate content, 2 studies (7,8) have reported that intake of dry carbohydrate (as starch or ground grain) — containing cat diets did not alter blood glucose concentrations. Although one might imagine that the method of processing, and the protein and fat content of the diet might influence the rate of carbohydrate digestion and metabolism, this was not found to be the case in these studies. However, one study showed the form of the carbohydrate to have an effect (8). In this study, carbohydrates included as starch did not significantly influence postprandial blood glucose concentrations (3.7 $\pm$ 0.7 mmol/L $\pm$ *s*, $n = 16$) compared with a carbohydrate-free diet (3.2 $\pm$ 0.8 mmol/L,

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n = 14) at 1, 3, or 6 h after feeding. Providing carbohydrate in the form of glucose led to a steep rise in blood glucose concentration 1 h after feeding (5.2 ± 0.7 mmol/L, n = 6), whereas providing carbohydrate in the form of sucrose induced a mild persistent hyperglycemia without marked postprandial changes (4.5 ± 0.5 mmol/L, n = 15). In commercial dry cat diets, carbohydrate is not commonly present as simple sugars, but as more complex starches and fibers.

A study by Appleton et al (9) identified differences in blood glucose in cats fed a diet containing equal quantities of ground corn and ground grain sorghum as the carbohydrate sources versus a diet containing only rice flour as the carbohydrate source. They found that the maximum incremental increase in glucose concentrations above baseline was significantly higher (P = 0.01) among cats fed the rice flour-based diet compared with those fed the ground sorghum/corn-based diet after the weight-maintenance phase. The extent to which these differences were due to differences in carbohydrate source or processing (flour versus grain) was not determined. Additionally, interpretation of both of these studies is limited by absence of food intake data.

With regard to the potential role of carbohydrates on obesity and type 2 diabetes mellitus (DM), one must consider a number of factors (10). Veterinary clinical nutritionists recognize at least 3 distinct groups of nutrition-related problems; diet induced, nutrient sensitive, and feeding related. Diet induced problems are those caused by a faulty diet and are treated by switching the patient to a diet that is known to be satisfactory. Nutrient sensitive problems are those where a disease or fault in the animal affects its response to a nutrient or ingredient. In these cases, a diet modified to accommodate the disease-induced nutritional limitations of the patient is recommended. Feeding related problems result from inappropriate feeding practices by the owner and are treated by client education (1).

Based on the available evidence, it does not appear that obesity or DM represent diet-induced diseases in cats. In 1994, an epidemiological investigation reported evidence for an increased risk of obesity in cats fed commercially available, high-carbohydrate, dry-expanded diets as opposed to commercially available, canned, high-fat diets (11). However, subsequent studies have not been able to replicate these results. Robertson (12) reported that the make-up of a cat's diet was not associated with its weight or weight category. He did find that overweight cats were more likely to be cross-bred, (odds ratio [OR] = 2.1), neutered (OR = 2.8), living in houses with only 1 or 2 cats (OR = 1.8), male (OR = 1.4), and predominantly confined inside a house (OR = 1.4). He concluded that obesity was influenced by a variety of factors related to the animal, the diet, and the management of the animal. In a recent population-based study of disease prevalence among 469 cats in the United States and Australia, 1 case of obesity (0.2%) and 7 cases of DM (1.5%) were identified. The prevalence of DM in cats may be compared to the population prevalence of ~5.5% in humans (13). One might have expected a significantly higher prevalence of DM in cats if the disorder was caused solely by an unsatisfactory diet. In contrast, a study of 8159 adult cats presented to veterinary practices identified 522 obese cats (6.4%) and 12 (2.3%) with DM.

In addition to the epidemiologic data, experimental studies have also found that carbohydrates per se may not be a major disease risk factor for cats. Thiess et al (14) recently conducted a short feeding trial of diets differing in carbohydrate and fat content on 6 intact and 6 neutered adult male cats. The "high carbohydrate" diet contained 9.7 g carbohydrate, 3.7 g fat, and 9 g protein/100 kcal, whereas the "high fat" diet contained 2.6 g carbohydrate, 5.9 g fat, and 10 g protein/100 kcal. They found that both diets were highly digestible, but that the cats fed the high fat diet showed a slightly elongated glucose clearance and reduced acute insulin response to glucose administration. These findings suggest diminished pancreatic insulin secretion, beta-cell responsiveness to glucose, or both in cats fed the high fat diet. Another recent study provided additional evidence that high dietary fat, but not carbohydrates, induced weight gain and increased insulin concentrations in cats, although plasma glucose concentrations were not affected by dietary fat percentage, neutering or weight gain (15).

Evidence is somewhat stronger for disorders having a nutrient-sensitive component in cats (10,12). Rand et al (10) summarized an impressive amount of evidence for roles that genetic and environmental factors play in feline DM. Evidence for genetic factors in feline DM included the overrepresentation of Australian-bred Burmese cats in the Australian cat population. Environmental risk factors in domestic or Burmese cats included advancing age, obesity, male gender, neutering, drug treatment, physical inactivity, and indoor confinement. However, even in Burmese cats, diet was not found to be a significant risk factor for development of DM (16). In addition, these environmental factors have also been linked to a variety of chronic diseases in cats (17). A more recent study by McCann et al (18) found that Burmese cats in an insured cat population in the United Kingdom were 3.7 times more likely to develop DM than were non-pedigree cats. They also reported a significantly increased risk ($P < 0.035$) of DM in cats fed either dry (OR = 1.1 to 4.5) or wet (OR = 1.2 to 7.4) diets than in those fed mixed diets. The authors concluded that, "DM is a complex disease with multiple factors interacting to result in increased risk of disease."

In a recent epidemiological study, Slingerland et al (19) collected information on dietary history and physical activity of 96 cats with DM and 192 matched controls using a telephone questionnaire. They found that the percentage of dry food in the diet was not significantly correlated with the development of DM (P = 0.29), whereas both indoor confinement (P = 0.002) and low physical activity (P = 0.004) were, further eroding support for the notion that the proportion of dry food in a cat's diet is an independent risk factor for the development of DM.

Feeding patterns may also be involved. As mentioned, cats naturally consume small multiple meals throughout the day, which limits meal-based intake of carbohydrates. Some cats, however, may be "greedy eaters," (10) and take in larger amounts of carbohydrate at each meal. Eating patterns may interact with diet composition. For example, effects of intake on circulating metabolite concentrations may be more significant for diets containing simple sugars (and possibly flours), than for those containing more complex starches and fibers.

Current published evidence thus does not support a direct role for diet in general, or carbohydrates in particular, on disease risk in domestic cats. However, available evidence does suggest that environmental and developmental factors may play a larger role in the development of chronic disease in cats than previously appreciated. If so, it may explain the focus on dry foods, which are very commonly fed to indoor-housed cats, and so might be expected to co-vary with other factors. Indoor-housed cats are kept under conditions similar to those of zoo animals; quite different from their natural environment, restricted in space, and dependent upon caregivers for all resources. These circumstances were recognized to affect the welfare of zoo animals, and zoos have responded in recent years with provision of species-specific resources that permit the animals to express their natural behavioral repertoires (20). These changes have improved zoo animal welfare (21) and suggest approaches that may be recommended by veterinarians caring for indoor-housed pets (17,22).

There is now considerable evidence that both genetic and environmental influences acting during early development also may influence vulnerability to disease later in life. The underlying mechanisms are thought to involve changes in gene expression to attempt to match individual responses to "predicted" environments. However, inaccurate predictions may increase the risk for obesity and DM (23–25); early experiences in cats that may be relevant in this context include orphaning and early trauma. These vulnerabilities may be unmasked by some later adverse event, such as an illness or injury, or by environments perceived as threatening. Given the strength of evidence in other species, future epidemiological studies might consider investigating the role of early experience on disease risk in cats. The data also suggest that cats may be yet another species to be affected by the global epidemic of obesity and DM (26). This epidemic, also known as 'diabesity,' is thought to be an unintended consequence of the pronounced changes in environment, lifestyle, and behavior that have accompanied globalization (26). In addition to humans and cats, evidence suggests that the epidemic has affected horses (27) and other species as well (Zimmet 2008, personal communication).

Until further evidence becomes available, there are a number of actions veterinarians can take now to reduce the risk of obesity and DM in cats including: educating owners about and providing resources that will create an enriched indoor environment to promote good welfare for their cats, including avoiding risks and expressing species-typical behaviors (22,28); recommending diets based on positive clinical experiences in maintaining long term health in cats; and recommending that cats be fed to a moderate body condition score (reminding clients to be particularly observant for increases in body condition score after neutering that may require adjustments in diet type, amount fed, or both). Available evidence suggests that the combination of these interventions may help reduce the risk of obesity and DM, as it has been shown to do for other chronic disorders in cats, more than the present narrow focus on carbohydrates in dry diets.

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